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ORIGINAL RESEARCH

Hypervigilance to Pain May Predict the Transition from Subacute to Chronic Back Pain: A Longitudinal Observational Study

Wen Zhang[1], Martin Löffler[1,2], Katrin Usai[1], Mina Mišić[1], Frauke Nees[1,3,*], Herta Flor[1,*]

[1]Department of Neuropsychology and Psychological Resilience Research, Research Group Learning and Brain Plasticity in Mental Disorders, Central Institute of Mental Health, Medical Faculty Mannheim, Heidelberg University, Mannheim, Germany; [2]Clinical Psychology, Department of Experimental Psychology, Heinrich Heine University Düsseldorf, Düsseldorf, Germany; [3]Institute of Medical Psychology and Medical Sociology, University Medical Centre Schleswig-Holstein, Kiel University, Kiel, Germany

*These authors contributed equally to this work

Correspondence: Wen Zhang, Department of Neuropsychology and Psychological Resilience Research, Research Group Learning and Brain Plasticity in Mental Disorders, Central Institute of Mental Health, Square J5, Mannheim, 68159, Germany, Tel +49 621 1703-6302, Fax +49 621 1703-6305, Email wen.zhang@zi-mannheim.de

Purpose: This study aimed to evaluate whether incorporating additional psychological and social risk factors, beyond those captured by the Örebro Musculoskeletal Pain Questionnaire (ÖMPQ), could enhance the prediction of the transition from subacute to chronic back pain.

Patients and Methods: Data of 75 patients with subacute back pain (SABP, 7–12 weeks) from a longitudinal observational study were analyzed. The ÖMPQ and additional emotional, cognitive, behavioural, and social factors were assessed at baseline. Pain severity and pain-related interference were assessed at baseline and six months later to evaluate chronicity. Principal component analysis reduced psychological variables into interpretable components. Pearson's correlation examined relationships between psychological factors, pain severity, and pain interference at baseline and 6-month follow-up. Factors linked to pain persistence were identified using best subsets regression and tested in multistage linear regression: (1) without baseline adjustment, (2) with baseline adjustment, and (3) assessing the effect of predictors from the previous steps on 6-month outcomes.

Results: The ÖMPQ and the Pain Vigilance and Awareness Questionnaire (PVAQ) scores significantly predicted pain severity, and the ÖMPQ, the Pain Behaviour Checklist (PBC), and PVAQ scores significantly predicted pain-related interference after six months. Multistage linear regression showed that PVAQ scores best predicted both pain severity (ß = 0.25, $p = 0.017$) and pain-related interference (ß = 0.33, $p = 0.002$) six months later, even after adjusting baseline pain levels.

Conclusion: The ÖMPQ score initially predicted pain persistence at six months, but its effect diminished after adjusting for baseline pain levels. In contrast, psychological risk factors such as pain hypervigilance and pain behaviors emerged as predictors of the pain severity or pain-related interference at six months. Pain hypervigilance became the strongest predictor of both pain severity and pain-related interference, regardless of initial pain, suggesting it as a target for early intervention.

Keywords: back pain, longitudinal studies, surveys and questionnaires, risk factors, anxiety, cognition

Introduction

Chronic back pain (CBP), particularly low back pain (LBP), is the leading cause of disability worldwide, with cases projected to rise from 619 million in 2020 to 843 million by 2050.[1,2] While most patients recover after acute pain onset, 26–32% transition to chronic back pain, resulting in long-term disability.[3,4] Identifying risk factors for this transition is important for reducing chronicity rates.

Over the past decades, extensive research has explored predictors of chronic pain. The biopsychosocial model emphasizes the role of psychological and social factors alongside biological ones in the development and persistence of chronic pain.[5–8] Neuroimaging studies revealed changes in brain areas involved in emotional processing and cognitive

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control, which can predict the transition to chronic back pain.[9–14] However, neuroimaging is costly and not routinely available in clinical settings, making psychological risk factor screening through questionnaires a practical and recommended alternative.[15–18]

Psychological risk factors in pain research cover a wide range of emotional, cognitive, and behavioural factors. Negative emotions such as anxiety, depression, stress are the earliest and most widely studied predictors of the chronicity in LBP.[19] Later studies found cognitive factors, including pain catastrophizing, attention, self-efficacy, beliefs and expectations, to be associated with the outcome of LBP.[20–24] Hypervigilance, characterized by heightened attention towards pain, may play a particularly significant role in the progression toward chronicity.[25–28] Closely tied to emotion and cognition, pain-related behaviour—especially fear-avoidance behaviour—is related to pain severity and disability in patients with chronic pain and may also drive the transition from acute to chronic pain.[29–31] Social factors—such as supportive and non-supportive responses from significant others (eg, spouses, caregivers), workplace conditions, cultural background, and economic status—further shape pain perception, coping with pain and disability, as well as overall quality of life in patients with back pain.[32–34]

Multidimensional questionnaires have been developed to improve screening and predict chronic pain by assessing multiple risk factors simultaneously. The Örebro Musculoskeletal Pain Questionnaire (ÖMPQ) is one of the most widely used instruments. It has been shown to be effective and reliable in predicting chronic pain, disability and absenteeism.[35] However it does not capture all psychological risk factors, particularly certain cognitive and social risk factors.[36] Identifying the key factors involved during the subacute stage of back pain (7 to 12 weeks post-onset) is important, as this period represents a critical window for the transition to chronicity, with pain characteristics differing significantly from those in the chronic stage.[37–39] Despite its importance, only a limited number of studies have investigated psychological and social risk factors of chronicity during this period.[40]

This longitudinal observational study investigates the transition from subacute to chronic back pain, using pain severity and interference at six months post-initial evaluation as indicators of chronicity. It aimed to cluster psychological and social risk factors based on their intercorrelations and to assess whether incorporating these factors improves prediction beyond those already captured by the ÖMPQ. We hypothesize that the inclusion of additional psychological and social risk factors would enhance the prediction of the transition from subacute to chronic back pain.

Materials and Methods

Design and Setting

The data analyzed in this study were collected from an ongoing longitudinal observational study within the Heidelberg Pain Consortium. Neuroimaging data from this study have been reported in prior publications.[33,41,42] Patients with SABP were followed longitudinally with a 6-month follow-up. Figure 1 outlines the study flowchart.

Ethical approval was obtained from the Ethics Committee of the Medical Faculty Mannheim of Heidelberg University (2014–584N-MA). The study was registered in the German Clinical Trials Register under ID DRKS00008835, and the registered information is available online (https://www.bfarm.de/). It was conducted in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki).[43] All participants provided written informed consent.

The study adheres to the STROBE guidelines for reporting observational longitudinal research (see Supplementary Material).[44]

Participants

Participants were recruited through the outpatient clinic of the Central Institute of Mental Health and collaborating pain clinics, flyers and newspaper announcements, online fora, and cooperation with general practitioners. All participants underwent comprehensive medical screening to exclude back pain due to specific underlying factors such as major injury, infection, systemic disease, or any known pathoanatomical conditions. The Structured Clinical Interview for DSM-IV (SCID-I) was used to assess comorbid mental disorders.[45]

Inclusion criteria for the SABP group included primary back pain—defined as back pain without a clear underlying cause, or pain (or its impact) disproportionate to any observable injury or disease.[46] The pain had to be located in the

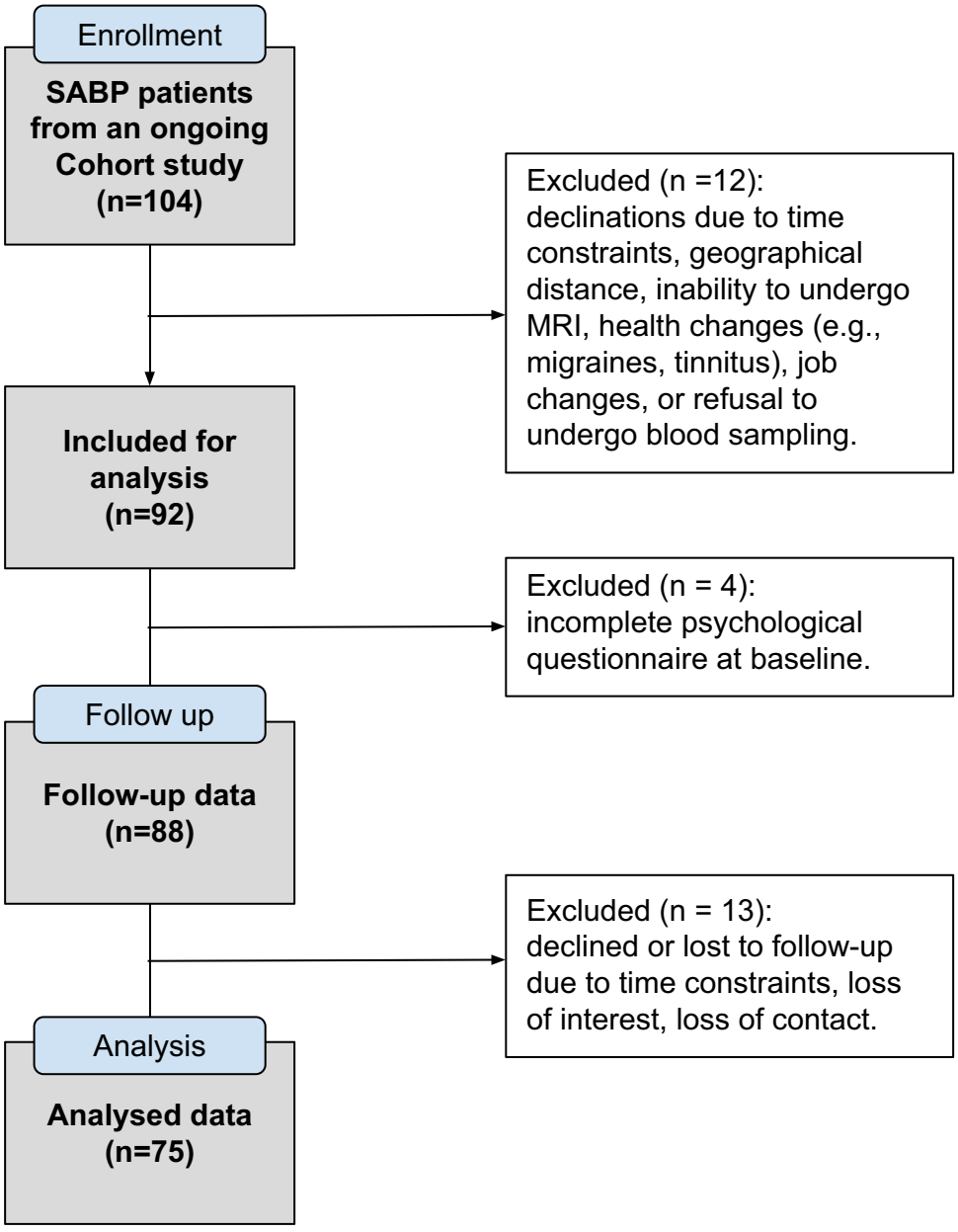

Figure 1 Flowchart of participant enrollment and exclusion in the study.

upper or lower back (or both) and had to last 7 to 12 weeks, as previously suggested in the literature.[39,47] Individuals with multiple short pain episodes lasting no more than three months were also included. Since this study also involved the examination of functional and structural magnetic resonance imaging, exclusion criteria were aligned with the overall cohort, excluding participants under 18 or over 70 years old, those with neurological complications, psychotic episodes, left-handedness, major illnesses, pregnancy, pacemakers, or metal implants.

The original sample size for patients with SABP for the ongoing prospective study was calculated based on an assumed mean conversion rate of 20%,[48] as observed in previous studies.[4,10] To ensure a minimum of 40 chronic pain cases after one year and thus adequate power, the study planned a sample size of N = 200 patients with SABP (power analysis based on G*Power 3.1.9.7, Heinrich-Heine-Universität Düsseldorf, Germany).[49,50] Despite significantly increasing recruitment efforts, only 92 patients with SABP were enrolled, likely due to the strict inclusion criteria and lower motivation among patients with subacute compared to patients with chronic pain to participate in research.

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Participants first underwent a telephone screening to gather demographic information and assess pain characteristics. They were then evaluated to identify factors that might influence pain or its chronicity using questionnaires, interviews, and clinical examinations. After six months, a follow-up telephone screening was conducted to reassess participants' pain status.

To evaluate the adequacy of the sample size for a linear multiple regression analysis, a post hoc power analysis was conducted using G*Power 3.1.9.7.[49,50] The analysis assessed the ability to detect a medium effect size ($f^2 = 0.15$) at a significance level of $\alpha = 0.05$, assuming 10 predictors and a total sample size of 92. The results indicated an achieved power of 0.98 (97.93%), exceeding the conventional threshold of 0.80 (80%) for adequate statistical power. Of the 92 enrolled patients with SABP, 88 completed baseline assessments, with follow-up data available for 75. Using the sample size of 75 in a recalculated post hoc power analysis, with other parameters unchanged, the actual achieved power was 0.95 (95.28%), confirming sufficient power to detect a medium effect size.

Measures

Assessment of Pain and Psychological Variables

The Örebro Musculoskeletal Pain Questionnaire (ÖMPQ) evaluates psychological risk factors for developing long-term pain-related disability in patients with musculoskeletal pain.[35,51] It comprises subscales on function, pain, psychological factors, fear avoidance, and miscellaneous items. The function subscale includes five items describing daily life activities and pain-related disability, while the pain subscale assesses current pain intensity, pain intensity during the past three months, pain sites, pain duration, and frequency. The sub-scale of psychological reactions evaluates anxiety, depression, perceived ability to cope with pain, perceived chance of the pain becoming persistent, and the chance of being able to work in six months. The fear-avoidance subscale assesses beliefs that pain increases with physical activity, that pain is a signal to stop activities, and that one should not work while in pain. The miscellaneous category includes items that cannot be grouped into one factor such as age, gender, working status, previous sick leave, heavy or monotonous work, and job satisfaction. The ÖMPQ has demonstrated moderate to good predictive value in numerous studies on chronic back pain with good validity and reliability.[52,53] Previously, a cutoff score of 105 and below indicated recovery with 95% accuracy, while a score of 130 and above indicated an 86% risk of patients developing a persistent back problem.[54] For this study, we used the German version of the original (long-form) ÖMPQ, which includes 25 questions, 21 of which are scored on a 10-point scale. It has demonstrated good reliability and validity.[35]

The West Haven-Yale Multidimensional Pain Inventory (MPI) was used in this study to evaluate pain severity and pain-related interference as outcome variables and responses of significant others to pain as psychological predictors.[55,56] It consists of three sections. The first section of the MPI encompasses five subscales: pain severity, pain-related interference, affective distress, social support, and life control. The second section includes responses of significant others to the patient's pain, with subscales for punishing responses, solicitous responses, and distracting responses. The third section assesses the patient's activity level with subscales for social and leisure activities, household activities, and outdoor activities that can be combined into a general activity level score. All MPI items are scored on a 6-point Likert Scale. The MPI has demonstrated high internal consistency and validity.[55]

The Hospital Anxiety and Depression Scale (HADS) measures anxiety and depression. It comprises 7 items in each subscale, with a 4-point scoring range.[57,58] It has demonstrated good reliability and validity.[57]

The German version of the Perceived Stress Scale (PSS) evaluates perceived stress.[59,60] The scale comprises 10 items, each rated on a 5-point Likert scale, with higher scores indicating higher levels of stress. It has been shown to be reliable and valid in measuring the level of stress.[60]

The German version of the Pain Behaviour Checklist (PBC) evaluates 11 pain-related behaviors, such as limping, grimacing, moaning, rigid posture, frequent changes in posture, verbal pain complaints, touching of painful areas, slowed movements, and refusal to perform activities due to pain, crying, and inactivity.[55,56] The item scores range from 0 to 2 (never, sometimes, and always). The German version of the PBC has demonstrated high reliability and validity.[61]

The Pain Vigilance and Awareness Questionnaire (PVAQ) is a 16-item measure assessing attention bias to pain in a questionnaire format, with each item scores ranging from 0 to 5 (never to always).[28,62] It has demonstrated good validity and reliability.[62]

The Fear of Pain Questionnaire III (FPQ III) evaluates fear of pain. It consists of three subscales with 30 items that assess severe, minor, and medical pain, respectively.[63,64] Each item is scored on a 5-point Likert Scale, with higher scores indicating greater fear. It FPQ III used in this study has demonstrated good validity and reliability.[64]

The Pain-Related Self-Statement Scale (PRSS) is an 18-item scale designed to assess catastrophizing and active coping in two subscales, each with 9 items.[65,66] The item scales range from 0 to 5 (nearly never to nearly always). The PRSS is a German equivalent to the Coping Strategy Questionnaire[66] and has shown good reliability and validity.[65]

All questionnaires were either originally developed in a German language version or were translated and validated for the German version.

Outcome Parameters

Pain severity and pain-related interference as assessed in the MPI were used as primary and secondary outcomes to assess back pain persistence over six months. These data were collected at baseline and at the 6-month follow-up.

Statistical Analysis

Statistical analyses were performed using RStudio Version 2024.04.2+764 (RStudio, PBC, Boston, MA, USA) with R 4.4.0 (© R Foundation for Statistical Computing, Vienna, Austria). The normality of all the data was assessed using the Shapiro–Wilk test. Normally distributed data are reported as means and standard deviations (SD), while non-normally distributed data are presented as medians with interquartile ranges (IQR).

Treatment of Outliers and Missing Values

In this longitudinal study, 4 out of 92 participants were excluded due to incomplete baseline assessments. An additional 13 participants (drop-out ratio 14.77%) declined or were lost to follow-up due to time constraints, loss of interest, and loss of contact. The final dataset included 75 patients with SABP. Outliers were identified as values with a $|z| \geq 3.29$ and were treated as missing values. The proportion of missing values across the 8 questionnaires and scales ranged from 9.3% to 22.7%. Little's MCAR test indicated that the missing data were completely at random (MCAR) with Chi-Square = 184.92, df = 175, p =0.289. We employed scale-level multidimensional imputations using the method of linear regression model.

Dimensionality Reduction in Psychological Factors

Principal component analysis (PCA) was employed to extract psychological components and reduce the 13 psychological variables in the SABP sample. We used the varimax rotation method and accepted factors with Cronbach's α above 0.5. Factor component scores were computed using Bartlett's approach.[67]

Correlation Analysis

Pearson correlation coefficients were used to examine the bivariate relationships between psychological risk factors—assessed through questionnaire, subscale, and factor score—as well as pain severity and pain-related interference at baseline and 6-month follow-up. The "pheatmap" package was employed to generate correlation heatmaps combined with hierarchical clustering.

Prediction Analysis

First, best subset regression identified the optimal model by selecting psychological factors validated through prior correlation analysis, along with age and gender. Variables from the model with the lowest Mallow's Cp and highest adjusted R^2 were chosen for further analysis.

Second, a multistage linear regression modeling procedure was used to predict the outcome variables and identify the best predictor. In the first step, predictors were tested without adjusting for baseline outcome values. In the second step, models were adjusted for these baseline values. Finally, significant predictors from the previous steps were evaluated for their effect on outcomes at the 6-month follow-up. A significance level of $\alpha = 0.05$ was used for all analyses.

Results

Participant Characteristics

Table 1 presents the demographic and clinical characteristics of the sample. The cohort was 65.33% female and 34.67% male, with a mean age of 36.00 years (SD = 13.24, range = 19–69). Twenty-eight cases (37%) reported a history of recurrent back pain. Additionally, six participants were taking ibuprofen, one was taking diclofenac, two were using paracetamol, one was using acetylsalicylic acid, and one was using a combination of acetylsalicylic acid, paracetamol, and caffeine. Table 2 shows the baseline scores of the psychological (sub)scales for all patients with SABP.

Dimensionality Reduction of Psychological Risk Factors

Table 3 presents the results of the principal component analysis (PCA) with a varimax rotation method and four fixed factors. The Kaiser–Meyer–Olkin's measure of sampling adequacy was 0.723, and Bartlett's Test of Sphericity was significant (chi-square (91) = 392.953, p <0.001), confirming the suitability for factor analysis. The four-factor solution explains 64.60% of the variance.

In the PCA analysis, the first factor (FAC1: negative emotion/stress) included MPI-Affective Distress, HADS, PSS, and MPI-Life Control (reversed). The second factor (FAC2: supportive social responses) comprised MPI-Solicitous Reactions, MPI-Distracting Reactions, and MPI-Support. PRSS-Active Coping (Coef. = 0.542), PVAQ (Coef. = 0.303), and MPI-General Activities (Coef. = 0.512) were kept as separate variables due to coefficients below the 0.6 threshold. Due to Cronbach's α of <0.5, the third (PBC and PRSS-Catastrophizing) and the fourth factors (MPI-Punishing Responses and FPQIII) were also independently analyzed in subsequent correlation and prediction analyses.

Table 1 Characteristics of the Sample with Subacute Back Pain (N=75)

Demographics	n	Mean (SD) or N (%)*
Age (years)	75	36.0 (13.24)
Gender-female/male	75	49 (65.33%)* /26 (34.67%)*
BMI[a]	74	24.76 (4.12)
Education time >12 years	73	53 (70.70)*
Employment-work	68	52 (76.47)*
Pain characteristics		
Upper back pain	75	12 (16)
Upper and middle back	75	1 (1.33)
Upper and lower back	75	8 (10.67)
Low back pain	75	23 (30.67)
Middle and back pain	75	1 (1.33)
Overall back pain	75	30 (40.00)
Back pain radiating down the leg[Δ]	32	3 (9.38)
Pain Severity (range 0 −10)	75	3.95 (1.67)
Pain-related Interference (range 0–10)	74	4.17 (2.51)
Pain duration in last 3 months (days)	73	29.55 (20.12)
Pain sites	67	2.42 (1.02)

Notes: [a]Body Index Mass; *Frequency (Percentage); [Δ] Including low back pain, middle and low back, upper and low back pain.

Table 2 Descriptive Statistics of Baseline Psychological (Sub)scales

Psychological (sub)Scales	Scale Range (min – max)	n	Mean (*SD*)	Median (*Q1 -Q3*)
ÖMPQ Sum	2–210	67	74.97 (20.42)	76 (60–90)
MPI-Pain severity	0–6	68	2.17 (1.09)	2.00 (1.33 −3.00)
MPI-Pain-related interference	0–6	68	1.65 (1.15)	1.40 (0.68–2.50)
MPI-Affective distress	0–6	68	2.55 (1.23)	2.33 (1.67–3.33)
MPI-Support	0–6	67	2.08 (1.60)	2.00 (0.83–3.00)
MPI-Life control	0–6	68	3.96 (1.21)	4.00 (3.00–5.00)
MPI-Punishing responses	0–6	64	0.70 (0.99)	0.17 (0–1.33)
MPI-Solicitous responses	0–6	64	2.61 (1.44)	2.80 (1.55–3.33)
MPI-Distracting responses	0–6	64	2.24 (1.55)	2.33 (1.00–3.33)
MPI-Social and leisure activities	0–6	67	3.30 (0.96)	3.25 (2.73–3.75)
MPI-Household activities	0–6	67	4.58 (1.08)	4.60 (3.80–5.60)
MPI-Outdoor activities	0–6	67	2.91 (1.27)	2.67 (2.00–3.90)
HADS	0–42	66	11.80 (7.46)	11.50 (7.00–16.00)
PSS	0–40	68	18.19 (7.48)	17.50 (12.00–23.00)
PBC	0–22	59	8.63 (3.35)	9.00 (6.00–11.00)
PVAQ	0–80	58	34.67 (10.23)	34.00 (29.00–40.00)
FPQ III	30–150	60	74.10 (16.84)	73.50 (64.00–83.25)
PRSS-Catastrophizing	0–5	59	1.19 (0.77)	1.11 (0.67–1.78)
PRSS-Active coping	0–5	59	3.10 (0.84)	3.11 (2.63–3.67)

Abbreviations: ÖMPQ, Örebro Musculoskeletal Pain Questionnaire; MPI, West Haven-Yale Multidimensional Pain Inventory; PRSS, Pain-related Self-statement Scales; PBC, Pain Behaviour Checklist; PVAQ, Pain Vigilance and Awareness; HADS, Hospital Anxiety and Depression Scale; PSS, the Perceived Stress Scale; FPQ III, Fear of Pain Questionnaire-III.

Table 3 Rotated Component Matrix of Principal Component Analysis (PCA)

	FAC 1	FAC 2	FAC 3	FAC4	Extraction
MPI-Affective distress	0.892				0.798
HAD	0.874				0.833
PSS	0.854				0.786
MPI-Life Control (reverse)	0.853				0.811
MPI-Solicitous Responses		0.825			0.741
MPI-Distracting reactions		0.750			0.670
MPI-Support		0.741			0.603
PRSS-Active Coping					0.365

(*Continued*)

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Table 3 (Continued).

	FAC 1	FAC 2	FAC 3	FAC4	Extraction
PRSS-Catastrophizing			0.760		0.655
PBC			0.725		0.592
MPI-General activities					0.601
PVAQ					0.446
MPI-Punishing Responses				−0.754	0.673
FPQIII				0.623	0.468
Eigenvalue	3.197	2.526	2.046	1.273	
% of Variance	22.837	18.043	14.615	9.093	
Cronbach's α	0.716	0.752	0.313	−0.030	

Notes: Absolute values of coefficients below 0.6 were suppressed to display.
Abbreviations: ÖMPQ, Örebro Musculoskeletal Pain Questionnaire; MPI, West Haven-Yale Multidimensional Pain Inventory; PRSS, Pain-related Self-Statement Scales; PBC, Pain Behaviour Checklist; PVAQ, Pain Vigilance and Awareness; HADS, Hospital Anxiety and Depression Scale; PSS, the Perceived Stress Scale; FPQ III, Fear of Pain Questionnaire-III; FAC 1, negative emotion/stress; FAC 2, supportive social responses.

Correlation Analysis

Figure 2 presents a correlation matrix of risk factors with hierarchical clustering. We followed widely cited guidelines for interpreting Pearson correlation coefficients, which suggest that $r \approx 0.1$ indicates a small association, $r \approx 0.3$ represents a moderate association, and $r \geq 0.5$ reflects a large association between variables.[68,69]

The ÖMPQ score and FAC1(negative emotion/stress) showed a moderate-to-large positive correlation ($r = 0.47$, $p < 0.001$) and were clustered together, suggesting they might share the aspect of emotional distress. Similarly, the PBS and PRSS-catastrophizing showed a moderate positive correlation ($r = 0.41$, $p < 0.001$) and clustered together, indicating a potential association between maladaptive cognitions and pain-related behaviors. These four variables—ÖMPQ score, FAC1, PBS, and PRSS-catastrophizing—were further intercorrelated: The ÖMPQ score and FAC1 showed moderate positive correlation with PBC ($r = 0.37$, $p = 0.001$ and $r = 0.27$, $p = 0.019$, respectively) and small-to-moderate positive correlations with PRSS-catastrophizing ($r = 0.28$, $p = 0.013$ and $r = 0.25$, $p = 0.033$, respectively). In addition, the ÖMPQ score had a moderate positive correlation with FPQIII ($r = 0.34$, $p = 0.003$), suggesting a meaningful relationship between emotional distress and fear of pain.

MPI-General Activities, FAC2 (Supportive Social Responses), PRSS-Active Coping formed a separate cluster, suggesting a potential link to the aspect of adaptive resilience. FAC2 demonstrated strong positive significant correlations with PRSS-Active Coping ($r = 0.54$, $p < 0.001$) and MPI-General Activity ($r = 0.51$, $p < 0.001$), while the direct correlation between PRSS-Active Coping and MPI-General Activity was not significant. In addition, MPI-Punishing Responses were separated from other clusters and showed no significant correlations with other risk factors.

Notably, PVAQ showed small-to-moderate positive correlations with FAC2 (Supportive Social Responses, $r = 0.30$, $p = 0.008$), PBS ($r = 0.25$, $p = 0.032$), and a moderate positive correlation with PRSS-catastrophizing ($r = 0.40$, $p < 0.001$). While FAC2 strongly positively associated with MPI-General Activities ($r = 0.51$, $p < 0.001$), PBS and PRSS-catastrophizing were moderately negatively associated with MPI-General Activities, with $r = -0.33$, $p = 0.004$, and $r = -0.31$, $p = 0.007$, respectively.

Table 4 shows the correlations between psychological risk factors and pain outcomes at baseline and follow-up. The results revealed strong associations between pain severity and pain-related interference (baseline: $r = 0.53$, $p < 0.001$; follow-up: $r = 0.61$, $p < 0.001$), indicating a slightly increasing link over time. Baseline pain severity moderately positively

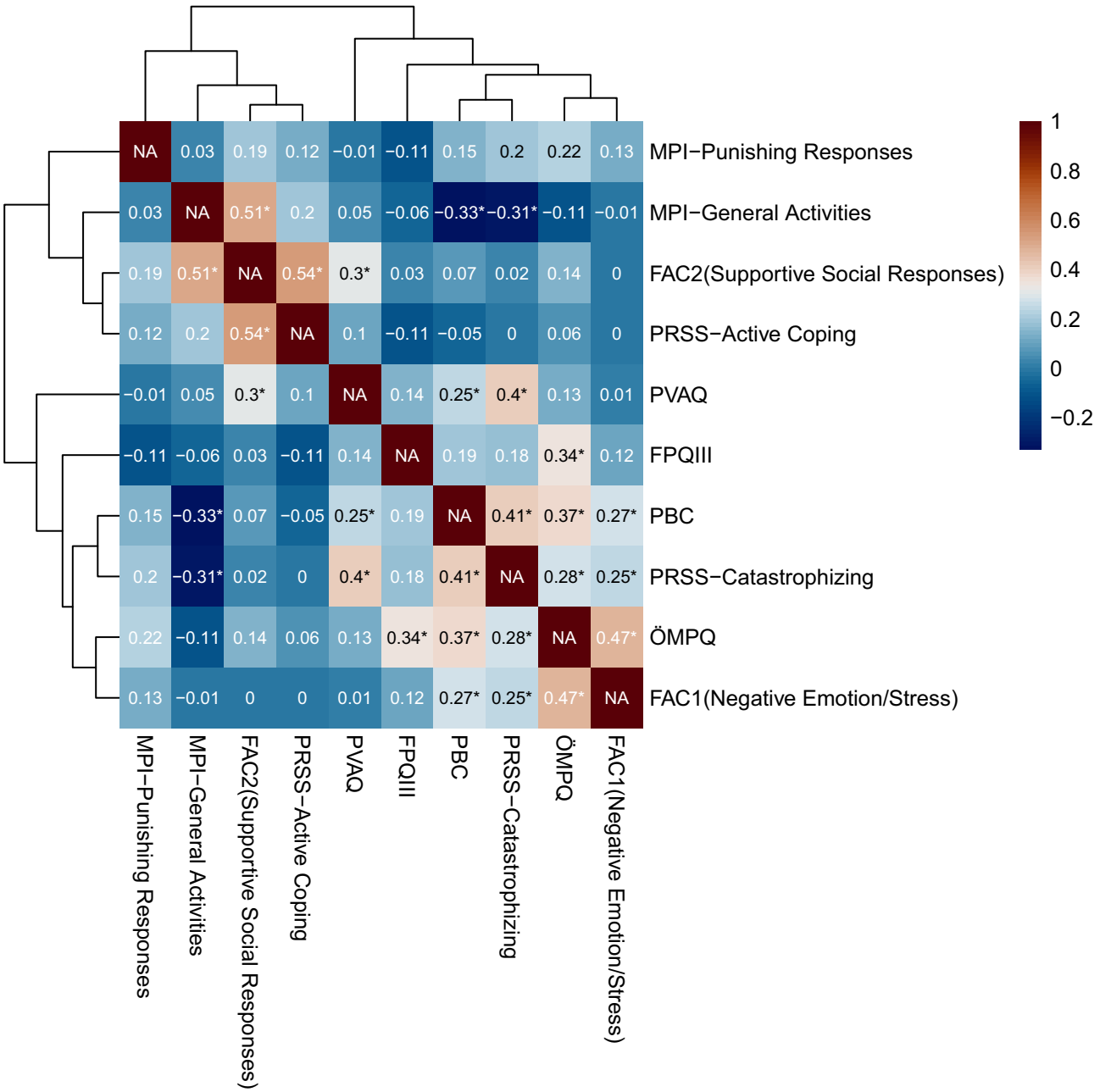

Figure 2 Correlation matrix heatmap combined with hierarchical clustering, illustrating relationships between psychological factors.
Note: Asterisks (*) indicate levels of statistical significance: $p < 0.05$.
Abbreviations: MPI, West Haven-Yale Multidimensional Pain Inventory; FAC 1 (Factor 1), negative emotion/stress; FAC 2 (Factor 2), supportive social responses; PRSS, Pain-related Self-Statement Scale; PVAQ: Pain Vigilance and Awareness; FPQ III: Fear of Pain Questionnaire-III; HADS: Hospital Anxiety and Depression Scale; PSS: the Perceived Stress Scale; PBC, Pain Behaviour Checklist; ÖMPQ, Örebro Musculoskeletal Pain Questionnaire.

correlated with the ÖMPQ score (r =0.38, p =0.001) and FAC2 (r =0.27, p =0.018), while pain-related interference showed moderate positive correlation with the ÖMPQ score (r =0.34, p =0.003) and small-to-moderate positive correlation with PBS (r =0.24, p =0.039). At 6-month follow-up, pain severity showed small but statistically significant positive correlation with the baseline ÖMPQ score (r =0.24, p =0.034) and a moderate correlation with the PVAQ (r =0.34, p =0.003). Pain-related interference showed moderate positive correlation with the ÖMPQ score (r =0.34, p =0.003), PBS (r =0.34, p =0.003), PVAQ (r =0.37, p =0.001), and small-to moderate correlations with PRSS-catastrophizing (r =0.30, p =0.010) and FAC1 (r =0.27, p =0.019). These factors associated with pain persistence were subsequently analyzed in the best subsets regression.

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Table 4 Correlations Between Psychological Risk Factors and Pain Outcomes at Baseline and Follow-up

	Baseline		Follow-Up	
	Pain Severity	**Pain-Related Interference**	**Pain Severity**	**Pain-Related Interference**
Pain Severity (baseline)	NA	**r = 0.53, *p* < 0.001**	**r = 0.46, *p* < 0.001**	**r = 0.33, *p* = 0.004**
Pain-related Interference (baseline)	**r = 0.53, *p* < 0.001**	NA	**r = 0.54, *p* < 0.001**	**r = 0.37, *p* = 0.001**
ÖMPQ	**r = 0.38, *p* = 0.001**	**r = 0.34, *p* = 0.003**	**r = 0.24, *p* = 0.034**	**r = 0.34, *p* = 0.003**
FAC1	r = 0.21, *p* = 0.072	r = 0.1, *p* = 0.37	r = 0.16, *p* = 0.167	**r = 0.27, *p* = 0.019**
FAC2	**r = 0.27, *p* = 0.018**	r = 0.22, *p* = 0.062	r = 0.14, *p* = 0.23	r = 0.09, *p* = 0.456
MPI-Punishing Responses	r = 0.18, *p* = 0.124	r = 0.09, *p* = 0.456	r = 0.05, *p* = 0.653	r = 0.06, *p* = 0.621
MPI-General Activities	r = 0.19, *p* = 0.095	r = −0.03, *p* = 0.827	r = 0, *p* = 0.998	r = −0.17, *p* = 0.147
PBS	r = 0.18, *p* = 0.119	**r = 0.24, *p* = 0.039**	r = 0.2, *p* = 0.078	**r = 0.34, *p* = 0.003**
PVAQ	r = 0.22, *p* = 0.064	r = 0.12, *p* = 0.295	**r = 0.34, *p* = 0.003**	**r = 0.37, *p* = 0.001**
FPQ III	r = 0.16, *p* = 0.173	r = 0.1, *p* = 0.399	r = −0.06, *p* = 0.62	r = 0.06, *p* = 0.63
PRSS-Active Coping	r = 0.11, *p* = 0.357	r = 0.07, *p* = 0.547	r = 0.06, *p* = 0.621	r = 0.03, *p* = 0.823
PRSS-Catastrophizing	r = 0.03, *p* = 0.796	r = 0.15, *p* = 0.204	r = 0.16, *p* = 0.181	**r = 0.3, *p* = 0.01**
Pain Severity (follow-up)	**r = 0.46, *p* < 0.001**	**r = 0.54, *p* < 0.001**	NA	**r = 0.61, *p* < 0.001**
Pain-related Interference (follow-up)	**r = 0.33, *p* = 0.004**	**r = 0.37, *p* = 0.001**	**r = 0.61, *p* < 0.001**	NA

Note: Bold values in the table indicate statistically significant correlations at $p<0.05$.
Abbreviations: ÖMPQ, Örebro Musculoskeletal Pain Questionnaire; MPI, West Haven-Yale Multidimensional Pain Inventory; PRSS, Pain-related Self-Statement Scale; PBC, Pain Behaviour Checklist; PVAQ: Pain Vigilance and Awareness; HADS: Hospital Anxiety and Depression Scale; PSS: the Perceived Stress Scale; FPQ III: Fear of Pain Questionnaire-III; FAC 1, negative emotion/stress; FAC 2, supportive social responses.

Regression Analysis

Table 5 summarizes the results of the best subset regression. For predicting pain severity after 6 months, Model 2 (comprising the ÖMPQ score and PVAQ) and Model 3 (comprising Age, the ÖMPQ score and PVAQ) exhibited the highest adjusted R^2 value (0.13). However, Model 2 demonstrated the lowest Mallows' Cp value (1.52), indicating a better model fit than Model 3. Regarding the prediction of pain-related interference after 6 months, Model 3 (comprising the ÖMPQ score, PBS, and PVAQ) exhibited the highest adjusted R^2 value (0.22) and lowest Mallows' Cp value (1.48). Hence, the ÖMPQ score and PVAQ from Model 2, as well as the ÖMPQ score, PBS, and PVAQ from Model 3, were employed in the subsequent multiple linear regression analysis.

Table 5 Summary of Best Subsets Regression

MI	**Predictors**	**R^2**	**R^2_{ad}**	**Mallows' Cp**
Independent variable: pain severity at 6 month				
1	ÖMPQ	0.11	0.10	3.00
2	ÖMPQ, PVAQ	0.16	0.13	1.52
3	Age, ÖMPQ, PVAQ	0.17	0.13	2.53
4	Age, ÖMPQ, PBC, PVAQ	0.17	0.12	4.16
5	Age, Gender, ÖMPQ, PVAQ	0.17	0.11	6.00

(*Continued*)

Table 5 (Continued).

MI	Predictors	R^2	R^2_{ad}	Mallows' Cp
Independent variable: pain-related interference at 6 month				
1	PVAQ	0.14	0.13	7.86
2	ÖMPQ, PVAQ	0.23	0.20	1.99
3	ÖMPQ, PBC, PVAQ	0.25	0.22	1.48
4	ÖMPQ, PBC, PVAQ, FAC1	0.27	0.23	2.22
5	Gender, ÖMPQ, PBC, PVAQ, FAC1	0.27	0.22	4.04
6	Gender, ÖMPQ, PBC, PVAQ, PRSS-Catastrophizing, FAC1	0.27	0.21	6.00
7	Age, Gender, ÖMPQ, PBC, PVAQ, PRSS-Catastrophizing, FAC1	0.27	0.20	8.00

Abbreviations: MI, model index; ÖMPQ, Örebro Musculoskeletal Pain Questionnaire; PRSS, Pain-related Self-Statement Scales; PBC, Pain Behaviour Checklist; PVAQ, Pain Vigilance and Awareness; FAC 1, negative emotion/stress.

To predict follow-up pain severity after 6 months, we first included the ÖMPQ score and PVAQ in model 1 and then conducted a multiple regression analysis that controlled for baseline pain severity in addition to these variables. Results are presented in Table 6. Model 1 explained 13% of the variance in follow-up pain severity (R^2 =0.16, $R^2_{adjusted}$ =0.13, F (2, 72) = 6.64, p =0.002), with the PVAQ (ß =0.31, t (72) =2.86, p =0.006), but not the ÖMPQ score (ß =0.21, t (72) =1.88, p =0.064), which was only marginally significant, being a significant predictor. An increase of one point in the PVAQ score was associated with an average increase of 0.06 points in the pain severity score (B =0.06, 95% CI [0.02, 0.10]) after 6 months. Model 2, adjusting for baseline pain severity, accounted for 24% of the variance in follow-up pain severity (R^2 =0.27, $R^2_{adjusted}$=0.24, F (3, 71) = 8.83, p <0.001). The PVAQ remained a significant predictor while the ÖMPQ score no longer approached significance (ß=0.07, t (71) =0.63, p=0.53). The final Model 3, which included baseline pain severity and the PVAQ, explained 25% of the variance in follow-up pain severity (R^2 =0.27, $R^2_{adjusted}$=0.25, F (2, 72) = 13.16, p<0.0001). The PVAQ continued to significantly predict follow-up pain severity (ß=0.25, t (72) =2.43, p=0.017), with each unit increase in the score corresponding to a 0.05-point increase in follow-up pain severity (B =0.05,

Table 6 Regression Coefficients for Predicting Follow-up Pain Severity

Variable	B	95% CI	ß	t	p
Model 1: R^2 = 0.16, R^2_{adj} = 0.13, F (2, 72) = 6.64, p =0.002					
ÖMPQ	0.02	[−.001, 0.04]	0.21	1.88	0.064
PVAQ	0.06	[0.02, 0.10]	0.31	2.86	0.006
Model 2: R^2 = 0.27, R^2_{adj} = 0.24, ΔR^2 = 0.001, F (3, 71) = 8.83, p < 0.0001					
Baseline pain severity	0.38	[0.16, 0.61]	0.38	3.36	0.001
ÖMPQ	0.01	[- 0.02, 0.03]	0.07	0.63	0.53
PVAQ	0.05	[0.01, 0.08]	0.25	2.39	0.02
Model 3: R^2= 0.27, R^2_{adj} = 0.25, ΔR^2 = 0.53, F (2, 72) = 13.16, p < 0.0001					
Baseline pain severity	0.41	[0.20, 0.62]	0.4	3.89	<0.001
PVAQ	0.05	[0.01, 0.08]	0.25	2.43	0.017

Abbreviations: CI, confidential interval for B; ÖMPQ, Örebro Musculoskeletal Pain Questionnaire; PVAQ, Pain Vigilance and Awareness.

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Table 7 Regression Coefficients for Predicting Follow-up Pain-Related Interference

Variable	B	95% CI	ß	t	p
Mode l: R^2 = 0.25, R^2_{adj} = 0.22, F (3, 71) = 8.03, p = 0.0001					
ÖMPQ	0.03	[0.001, 0.05]	0.23	2.10	0.039
PBC	0.12	[−0.03, 0.27]	0.18	1.61	0.112
PVAQ	0.07	[0.02, 0.11]	0.30	2.83	0.006
Model 2: R^2 = 0.29, R^2_{adj} = 0.26, ΔR^2 = 0.02, F (3, 71) = 9.55, p < 0.001					
Baseline pain-related interference	0.22	[0.04, 0.39]	0.26	2.47	0.016
ÖMPQ	0.03	[−.0005, 0.05]	0.21	1.96	0.054
PVAQ	0.07	[0.03, 0.11]	0.32	3.12	0.003
Model 3: R^2 = 0.25, R^2_{adj} = 0.23, ΔR^2 = 0.05, F (2, 72) = 11.94, p < 0.001					
Baseline pain-related interference	0.27	[0.10, 0.44]	0.33	3.23	0.002
PVAQ	0.07	[0.03, 0.12]	0.33	3.24	0.002

Abbreviations: CI, confidential interval for B; ÖMPQ, Örebro Musculoskeletal Pain Questionnaire; PBC, Pain Behaviour Checklist; PVAQ, Pain Vigilance and Awareness.

95% CI [0.01, 0.08]), while each unit increase in the score of baseline pain severity corresponding to a 0.41-point increase in follow-up pain severity score (B =0.41, 95% CI [0.20, 0.62]).

The same multistage linear regression analysis was used to predict pain-related interference at the 6-month follow-up, with the ÖMPQ score, PBS and the PVAQ included as predictors. Results are presented in Table 7. Model 1 explained 22% of the variance of pain-related interference at the follow-up measurement (R^2 =0.25, $R^2_{adjusted}$=0.22, F (3, 71) = 8.03, p =0.0001). Both the ÖMPQ score (ß=0.23, t (73) = 2.10, p=0.039) and PVAQ (ß=0.30, t (73) = 2.83, p=0.006) were significant predictors, while the PBS failed to significantly predict pain-related interference at the follow-up measurement (ß=0.18, t (71) =1.631 p=0.11). One unit increase in the ÖMPQ score corresponded to a 0.03 rise in pain-related interference (B=0.03, 95% CI [0.001, 0.05]), while one unit increase in the PVAQ score resulted in a 0.07-point increase (B=0.07, 95% CI [0.02, 0.11]). Model 2, after controlling for baseline pain-related interference, explained 26% of the variance of pain-related interference at the follow-up measurement (R^2 =0.29, $R^2_{adjusted}$=0.26, F (3, 71) = 9.55, p <0.001). In this model, only PVAQ remained a significant predictor (ß=0.32, t (71) = 3.12, p=0.003), while the ÖMPQ score was only marginally significant in predicting pain-related interference at the follow-up measurement after controlling for pain-related interference at baseline (ß=0.21, t (71) = 1.96, p=0.054). Model 3 included both baseline pain-related interference and PVAQ. It explained 23% of the variance in follow-up pain-related interference (R^2 =0.25, $R^2_{adjusted}$=0.23, F (2, 72) = 11.94, p <0.001). The PVAQ was a significant predictor of pain-related interference at the follow-up measurement in Model 3 (ß=0.33, t (72) = 3.24, p =0.002), with each unit of increase in the PVAQ score leading to a 0.07-point increase in follow-up pain-related interference (B =0.07, 95% CI [0.03, 0.12]), while each unit increase in the score of baseline pain-related interference to a 0.27-point increase in follow-up pain-related interference (B =0.27, 95% CI [0.10, 0.44]).

Discussion

We evaluated whether incorporating additional psychological and social risk factors—beyond those captured by the ÖMPQ—could improve the accuracy of predicting the transition from subacute to chronic back pain. Our findings extend the results of previous studies by showing that additional factors such as emotional distress, hypervigilance and pain-related behavior, are associated with the development of chronic pain.

Correlation Patterns of Psychological Risk Factors and the Dual Role of Pain Hypervigilance

Correlation analysis and hierarchical clustering identified two distinct patterns of psychological and social risk factors included in this study. The first pattern, representing maladaptation, included negative emotions, cognitions, and behaviors, which clustered together (ÖMPQ, FAC1, PRSS-catastrophizing, and PBS) as mutually reinforcing factors. The second pattern, reflecting adaptive resilience, included social support, positive coping, and engagement in activities (FAC2, PRSS-Active Coping, and MPI-General Activity). Interestingly, pain hypervigilance was correlated with both patterns and could influence general activity levels in opposite ways: it can encourage activity through the positive correlation with social supports (FAC2), yet discourage it through its association with negative cognitive (PRSS-catastrophizing) and behavioral (PBS) factors. Furthermore, the association with supportive social responses (FAC2) suggests that pain hypervigilance may also serve a communicative function.

Pain hypervigilance, which can either promote or reduce activity levels at the subacute stage, has been identified as a possible predictor of chronic back pain in subsequent regression analysis. Initially, hypervigilance may be a modifiable factor; however, its association with catastrophizing and pain-related behaviors suggests that heightened pain awareness, combined with negative cognition-behavior pattern, might lead to activity avoidance, ultimately as pain persists, it may shift into dominant maladaptive patterns that intensify pain severity, interference, disability and finally lead to pain chronicity.

This dual role of pain hypervigilance underscores the potential of interventions, such as attention management and cognitive-behavioral therapy (CBT),[70] to enhance the adaptive aspects of hypervigilance—such as fostering social support and active coping—while reducing maladaptive patterns linked to emotional distress and catastrophizing. Recognizing hypervigilance as a risk factor is important for clinicians and researchers, as inadequate awareness or miscommunication may contribute to nocebo effects, ultimately reducing treatment efficacy for musculoskeletal pain.[71,72]

Prediction of Pain Progression

The ÖMPQ is a multidimensional questionnaire that focuses on screening psychological risk factors for chronic pain development, particularly emotional distress components like depression, anxiety, and fear avoidance. In this study, we intended to test if the use of additional psychological variables such as pain hypervigilance, catastrophizing or pain behaviors beyond the ÖMPQ could provide predictive accuracy for pain progression at the subacute stage. The ÖMPQ score alone as well as with the scores of PBC and PVAQ at this stage predicted pain persistence after 6 months. However, the effect of these predictors diminished after baseline adjustment, and only the PVAQ remained predictive of chronicity. One possible explanation is the moderate positive correlation between the ÖMPQ and baseline pain severity, as 11 of its 21 items assess pain characteristics, suggesting that its predictive effects may partly reflect autoregressive influences.

Alternatively, the psychological risk factors identified by the ÖMPQ may only impact pain progression within certain time frames rather than during the entire subacute stage. Correlation analysis revealed the shifts in psychological risk factors over time. At baseline, pain severity was associated with the ÖMPQ score and social support (FAC2), suggesting a link to increased emotional distress and social support needs at the subacute stage. By follow-up, the ÖMPQ score remained consistently correlated with pain severity, while pain hypervigilance, as measured by PVAQ, emerged as a relevant factor and the impact of social support diminished, suggesting the intensified influence of cognitive factors. Pain-related interference showed consistent correlations with the ÖMPQ score and pain-related behaviors (PBS) from baseline to follow-up, indicating stable contributions of these factors. At follow-up, additional correlations emerged with catastrophizing (PRSS), negative emotions and stress (FAC1), and pain hypervigilance (PVAQ), suggesting full involvement of negative emotions and an increased role of maladaptive cognitive factors as back pain became chronic.

These findings suggest that psychological factors may exert their effects within specific time frames or shift in influence as pain progresses. While the impact of maladaptive cognitions intensifies as SABP progresses, the ÖMPQ's primary focus on emotional distress associates it more closely with pain severity in the subacute stages.

Hypervigilance to Pain Emerged as a Potential Predictor at the Subacute Stage for Pain Chronicity

Hypervigilance to pain emerged as a strongest predictor at the subacute stage for the chronicity of back pain. This aligns with evidence linking pain hypervigilance to higher pain severity.[73–76]

Attention plays a direct role in modulating sensation. Animal and human neuroimaging studies have shown that neural activity of somatosensory neurons could be modulated by attention, with focused attention amplifying signals in pain perception and distraction reducing it.[77,78] Clinical observations align with these findings: concentrating on pain tends to amplify the perception of its intensity, whereas distracting or redirecting attention can alleviate it.[27,79,80] However, clinical studies also found that individual responses to attention manipulation vary, with some participants experiencing no or paradoxical pain reductions when using the same attention manipulation strategies.[75,81,82] These differences may stem from individual variations in attention, where some individuals are naturally more vigilant to pain-related stimuli.[83] This hypervigilance is also associated with an increased risk of developing chronic pain.[84]

In patients with chronic pain, attention bias to pain has been confirmed by a meta-analysis study.[85] Attention bias is generally divided into avoidance and hypervigilance from the direction of the bias of attention.[86] Hypervigilance to pain correlates with higher pain intensity, emotional distress, and psychological disability, even when controlling for baseline pain intensity and demographic factors.[28] In previous studies, vigilance exacerbated pain by intensifying pain sensations and reducing engagement in productive activities.[28,84]

Attention Bias Modification (ABM), an intervention aimed at altering attentional deployment to symptom-relevant emotionally salient stimuli, has shown potential in improving pain outcomes by raising experimental pain thresholds in healthy participants.[87] Clinically, ABM has demonstrated effectiveness in reducing pain intensity, disability and unfavorable psychological consequences in patients with chronic pain.[88] This suggests that heightened vigilance to pain may contribute to chronicity by influencing an individual's pain perception.

Our study highlights the potential predictive role of hypervigilance to pain in the transition from subacute to chronic back pain, suggesting that hypervigilance during this period may significantly influence pain progression. Therefore, attention management at the subacute stage could be an effective strategy for preventing the development of chronic back pain.

Strengths and Limitations

This study has several strengths. First, it addresses a critical gap in the literature by focusing on the subacute stage of back pain, a period that represents a key window for intervention to prevent chronicity. Second, the assessment of psychological and social risk factors is extensive. Third, the use of robust statistical methods ensures methodological rigor. Especially, the use of correlations and hierarchical clustering revealed two distinct resilience patterns—maladaptive and adaptive—and their correlations with pain hypervigilance, offering a novel perspective on the predictive value of hypervigilance. Fourth, the longitudinal design provides valuable insights into the dynamic nature of risk factors over time. Finally, the study's findings have significant clinical relevance, offering actionable insights for developing targeted early interventions to reduce the burden of chronic back pain.

This study also has several limitations. First, our cohort consisted of a carefully selected sample of SABP participants with primary pain. This, along with the relatively small sample size, may limit the generalizability of our findings and reduce the feasibility of more detailed analyses. Second, the strict inclusion criteria may have introduced selection bias. Third, there was an imbalance in the gender distribution of SABP participants in our study, with approximately two times more women than men. This ratio is slightly higher than 1.4, which was reported in a large-scale randomized cohort study in patients with acute low back pain,[4] but lower than 2.6 observed in a cross-sectional study on chronic pain.[89] This discrepancy may stem from the higher prevalence of pain in women compared to men and potential gender-based differences in clinical trial participation.[90–92] Due to the limited sample size in our study, gender matching was not feasible. Although the best subset regression analysis indicated that gender had no significant effect on the outcome variables, this imbalance may still limit the applicability of our results to male participants. Fourth, baseline data indicated that participants experienced mild to moderate pain severity. Since pain and psychological risk factors are interrelated, different risk factors may emerge in individuals with more severe pain during the subacute phase. Fifth, our analysis lacks post-hoc correction. Recommendations for post-hoc correction are inconsistent, and using different correction methods can yield conflicting p-value results; additionally, post-hoc correction itself may increase Type II errors.[93] Given our study's exploratory nature and focus on managing Type I errors, we choose not to apply post-hoc correction and have interpreted the findings with caution. Lastly, in this study, we have selectively focused on psychological and social variables related to chronicity, without addressing other potentially relevant predictors such as

personality traits, central sensitization, brain imaging characteristics, or other biological factors.[94] Future studies should evaluate the relative contribution of these determinants to chronicity.

Conclusion

At the subacute stage, the ÖMPQ score, alone or with PBC and PVAQ, predicted pain persistence at six months but lost significance after the adjustment of baseline pain, likely due to overlap with pain severity. Our data suggest that maladaptive cognitions (eg, pain hypervigilance, catastrophizing) gain influence as pain transitions to chronicity.

Pain hypervigilance may have a dual role at the subacute stage, fostering social support through increased activity but also limiting it through its association with catastrophizing and pain-related behaviors. As pain advanced, individuals with SABP who exhibited hypervigilance were more likely to report greater pain severity and interference six months later.

These findings highlight the role and predictive value of pain hypervigilance in the transition from subacute to chronic back pain. Early interventions targeting hypervigilance may help to prevent chronicity by enhancing adaptive coping while reducing maladaptive patterns tied to negative emotions, cognitions and behaviors during the subacute stage. Future longitudinal studies should incorporate behavioral measures of attentional bias to confirm these findings and to clarify the mechanisms linking hypervigilance to pain chronicity.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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